

8. Increase of interval is never accompanied by a fall in the summated contraction, provided the second stimulus falls before or within the period of "ascending energy."

9. Having regard to the causes of variation given above, we do not find a sudden increase in summative effect to be constant at any one period of increment of interval.—*Journal of Physiology*, vol. vi., No. 3.

THE NATURE OF NERVE-FORCE.—Dr. Bowditch has made some experiments upon this point. The principal data of which account must be taken in every proposed theory of nerve-force, are :

1. The transmission of a stimulus along the nerve with undiminished intensity.

2. The exhaustion of the nerve by continued stimulation.

Upon an etherized cat the sciatic was divided near the sacrum and placed upon a pair of shielded electrodes of an induction machine.

The animal received then a dose of curare sufficient to prevent muscular contractions, and the irritation of the nerve was steadily maintained while the animal was kept alive. In the course of about two hours the curare was so far eliminated that the stimulation of the nerve which had been previously without effect began to produce muscular twitches which, as the elimination of the drug progressed, became more frequent and more violent. These experiments support the theory that nerve-force is transmitted by some sort of a molecular vibration after the manner of light or electricity. An argument of a negative character in favor of this view may be found in the absence of satisfactory evidence of chemical change or of heat production associated with the activity of the nerve, showing that no great amount of potential energy is set free in the process.

Physiologists have long been in the habit of comparing nerves to telegraph wires, since they seem to be indifferent conductors transmitting impulses equally well in both directions. It would appear from these experiments that the absence of fatigue in consequence of activity is another very interesting point of resemblance.—*Journal of Physiology*, vol. vi., No. 3.

ISAAC OTT, M.D.

c.—GENERAL PATHOLOGY OF THE NERVOUS SYSTEM.

ANALYSIS OF THE NERVE-PHENOMENA IN ANÆSTHETIC LEPROSY.—W. Allen Sturge, M.D., in a paper (*Brain*, April, 1885) based upon the careful study of a case of non-tuberculated anæsthetic leprosy, reaches the following conclusions in answer to certain queries which he propounds—namely, (1) Is the lesion *central*,—i. e., in the brain or spinal cord ; or is it *peripheral*,—i. e., in the nerves ? (2) If peripheral, is it (a) in the trunks of the nerves ? (b) in the finer nerve branches ? or (c) in the peripheral

termination of the nerves? He concludes that both the sensory and motor phenomena are dependent upon a peripheral-nerve lesion, but that the former result primarily from implication of the terminal sensory filaments without relation to the distribution from any particular nerve-trunk, while the motor impairment depends upon the involvement of a mixed nerve-trunk. He was led to this view from the fact that the anæsthesia is superficial, not deep, and does not correspond to the distribution of one nerve, but creeps on, involving areas of skin without reference to the nerve-supply. On the other hand, the muscular atrophy occurs in the distribution of definite nerve-trunks, and not in muscles associated functionally to produce co-ordinate movements, as in certain spinal-cord lesions.

He explains these diversely located lesions, as regards sensory and motor phenomena, as follows: Pointing out the fact that the leprous condition has a strong tendency—in the non-tuberculated variety, at any rate—to advance by continuity of tissue, that the skin and nerves have a special tendency to be attacked, he infers that the nervous affection begins at the peripheral extremities of the cutaneous nerves, and, creeping up the nerve fibres, extends eventually to the main nerve-trunks. As the nerve disease has been shown to consist mainly of a cellular new-formation, accompanied by some hypertrophy of the connective tissue binding the fibres together, those fibres not already injured by the new growth become compressed and ultimately destroyed. As the disease begins in the peripheral extremities of these latter nerves, the motor nerves will not be affected until it has passed up the cutaneous branches to the main trunk, from which the motor nerve is given off. When this has happened, a whole group of muscles will be affected simultaneously—a group deriving its nerve supply from a common origin. He believes that muscle directly under the skin, as in the face, may be affected directly by contiguity from the skin.

He suggests the following points for investigation in future cases: (1) In cases of tuberculated and anæsthetic leprosy (typical tuberculated leprosy is non-anæsthetic), to notice the difference between the eruption of parts of the skin where sensation is healthy, and that of patches of anæsthetic skin. With a view to proving the secondary influence of nerve disease in checking the morbid growth in the skin. (2) Is anæsthesia always preceded by modification in skin nutrition? With a view to proving the independence of the nerve disease. (3) Does the primary invasion of anæsthesia ever give rise to patches of numbness corresponding to the supply of any named nerve? With a view to clearing up the question whether nerve-trunks are ever subjected to direct attack. (4) Does muscular atrophy ever take place without being preceded by anæsthesia? With a view to proving whether ultimate motor branches may be primarily affected, in a way similar to ultimate sensory branches. (5) When muscular atrophy has taken place, to make a detailed examination of the various

groups of muscles. With a view to confirming or confuting the conclusions arrived at above, in reference to the mode in which their atrophy is produced. (6) An investigation of the deep sensibility of parts beneath the anæsthetic skin, and a comparison of the deep sensibility of healthy muscles as compared with that of atrophied muscles.

A CASE OF PERFORATING TUBERCULOSIS OF SKULL WITH CEREBRAL SYMPTOMS.—Dr. Walter Edmunds reports (*Brain*, April, 1880) the case of a boy, æt. fourteen, who, after an attack of peritonitis, which was thought to be tubercular, developed a cold abscess in the scalp, over the left parietal bone, accompanied by much headache. It contained thick pus, and was twice aspirated. Six months later, numbness of the right arm and hand occurred; four months later, the abscess re-formed, and he had a fit, in which he felt giddy, fell, became unconscious, foamed at the mouth, bit his tongue, urinated involuntarily, had convulsive movements in both legs, with the right arm clinched and drawn up. Slight paresis of the right face and leg, with more marked paresis of the right hand, was found, with normal sensation, normal temperature and reflexes. No visual disturbance nor headache; both optic discs were slightly swollen, their images blurred; veins knotted, with white lines along the vessels. The abscess was laid freely open, and a portion of diseased bone, seven eighths by five eighths of an inch, was removed, comprising the entire thickness of the skull. It had compressed the brain. Under iodoform dressing the case progressed well, but there was some suppuration with loss of substance on the surface of the brain. He had one fit two weeks later, another eight weeks later, with general convulsions, head turned to the right. In about four months he was sufficiently well to get up and walk about. Weakness of the right arm and hand remained, and an opening in the scalp and skull, through which the brain could be seen. It was covered by a silver plate. The inflammation of the optic discs subsided, the sight remaining not quite normal. The opening in the skull was half an inch in front of the fissure of Roland, but as the superficial ulceration extended backward a short distance from the opening, the middle of the ascending frontal and ascending parietal convolutions were affected.

ACUTE OPTIC NEURITIS ASSOCIATED WITH ACUTE MYELITIS.—Sharkey and Lawford (*Ophthal. Soc. Trans.*, London).—A girl, aged seventeen, previously in good health, rapidly lost her sight, so that in four days she was quite blind, without other prominent symptoms. Well-marked double optic neuritis was found to be present a month afterward. Symptoms of paralysis and loss of sensation in the lower extremities supervened. About three weeks afterward the patient died of symptoms of peritonitis—that